In the claims:

Amend claims 3-9, 14, 16, 17, 24, 25, 27, 28, and 36-40 as follows:

-- 3. (Amended) The method of claim 1, wherein the TAK1 or the TAB1 is linked to a support.

4. (Amended) The method of claim 1, wherein a label is attached to the TAK1 or the TAB1 and wherein the binding is detected by detecting or measuring the label.

5. (Amended) The method of claim 1, wherein the binding is detected by detecting or measuring the TAB1 bound to the TAK1 with a primary antibody against TAB1 or a primary antibody against the peptide fused with the TAB1.

6. (Amended) The method of claim 1, wherein the binding is detected by detecting or measuring the TAK1 bound to the TAB1 with a primary antibody against TAK1 or a primary antibody against the peptide fused with the TAK1.

7. (Amended) The method of claim 1, wherein the binding is detected by detecting or measuring the TAB1 bound to the TAK1 with a primary antibody against the TAB1 or a primary antibody against the peptide fused with TAB1, and a secondary antibody against the primary antibody.

8. (Amended) The method of claim 1, wherein the binding is detected by detecting or measuring the TAK1 bound to the TAB1 with a primary antibody against TAK1 or a primary antibody against the peptide fused with the TAK1, and a secondary antibody against the primary antibody.

9. (Amended) The method of claim 5, wherein the primary antibody or the secondary antibody is labeled with radioisotope, enzyme, or fluorescent substance.

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14. (Amended) The method of claim 12, wherein a substrate for the TAK1 is added and wherein the phosphorylation of the substrate by the TAK1 is detected.

16. (Amended) The method of claim 12, wherein the TAK1 is fused with a peptide.

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17. (Amended) The method of claim 12, wherein the TAK1 is linked to a support.

24. (Amended) The method of claim 20, wherein the reporter gene is luciferase, chloramphenicol acetyltransferase, green fluorescent protein, or β-galactosidase.

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25. (Amended) The method of claim 18, wherein an inflammatory stimulus is given to cells and wherein the biological activity transduced through TAK1 or through TAK1 and TAB1 is detected and/or measured.

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27. (Amended) The method of claim 1, wherein the inflammatory cytokine is IL-1, TNF, IL-10, or IL-6.

28. (Amended) A compound for inhibiting signal transduction through inflammatory cytokines, the compound that can be isolated by the method of claim 1.

36. (Amended) The pharmaceutical composition of claim 33, wherein the pharmaceutical composition is an anti-inflammatory agent.

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37. (Amended) The pharmaceutical composition of claim 33, wherein the compound is a compound inhibiting binding between TAK1 and TAB1.

38. (Amended) The pharmaceutical composition of claim 33, wherein the compound is a compound inhibiting phosphorylation by TAK1.

39. (Amended) The pharmaceutical composition of claim 33, wherein the compound is a compound that can be isolated by the steps of:

(a) contacting a test sample with TAK1 and TAB1;

(b) detecting binding between the TAK1 and the TAB1; and

(c) selecting a compound inhibiting the binding.

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40. (Amended) The pharmaceutical composition of claim 33, wherein the inflammatory cytokine is IL-1, TNF, IL-10, or IL-6. --